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NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2	Dec 17	The CA Lexicon available in the CAPLUS and CA files
NEWS	3	Feb 06	Engineering Information Encompass files have new names
NEWS	4	Feb 16	TOXLINE no longer being updated
NEWS	5	Apr 23	Search Derwent WPINDEX by chemical structure
NEWS	6	Apr 23	PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
NEWS	7	May 07	DGENE Reload
NEWS	8	Jun 20	Published patent applications (A1) are now in USPATFULL
NEWS	9	JUL 13	New SDI alert frequency now available in Derwent's DWPI and DPCI

NEWS EXPRESS	July 11 CURRENT WINDOWS VERSION IS V6.0b, CURRENT MACINTOSH VERSION IS V5.0C (ENG) AND V5.0JB (JP), AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2001
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
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FILE 'HOME' ENTERED AT 17:13:00 ON 06 AUG 2001

=> file medline

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.15	0.15

FILE 'MEDLINE' ENTERED AT 17:13:09 ON 06 AUG 2001

FILE LAST UPDATED: 30 JUL 2001 (20010730/UP). FILE COVERS 1958 TO DATE.

On April 22, 2001, MEDLINE was reloaded. See HELP RLOAD for details.

MEDLINE now contains new records from the former NLM HEALTH STAR database. These records have an Entry Date and Update Date of 20010223.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2001 vocabulary. Enter HELP THESAURUS for details.

The OLDMEDLINE file segment now contains data from 1958 through 1965. Enter HELP CONTENT for details.

Left, right, and simultaneous left and right truncation are available in the Basic Index. See HELP SFIELDS for details.

THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE SUBSTANCE IDENTIFICATION.

```
=> s (CD40 or CD154) (w) fusion
          3269 CD40
           277 CD154
         86517 FUSION
          6354 FUSIONS
         89245 FUSION
                (FUSION OR FUSIONS)
L1           3 (CD40 OR CD154) (W) FUSION

=> d l1 ibib abs

L1   ANSWER 1 OF 3     MEDLINE
ACCESSION NUMBER:    2001209684     MEDLINE
DOCUMENT NUMBER:     21195371   PubMed ID: 11298824
TITLE:               Rewiring of CD40 is necessary for delivery of rescue
                     signals to B cells in germinal centres and subsequent
entry
                     into the memory pool.
AUTHOR:              Siepmann K; Skok J; van Essen D; Harnett M; Gray D
CORPORATE SOURCE:    Department of Immunology, Imperial College School of
                     Medicine, Hammersmith Hospital, London, UK.
SOURCE:              IMMUNOLOGY, (2001 Mar) 102 (3) 263-72.
                     Journal code: GH7; 0374672. ISSN: 0019-2805.
PUB. COUNTRY:        England: United Kingdom
                     Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:            English
FILE SEGMENT:        Priority Journals
ENTRY MONTH:         200105
ENTRY DATE:          Entered STN: 20010517
                     Last Updated on STN: 20010517
                     Entered Medline: 20010510
```

AB Memory B-cell development is impaired by in vivo blockade of the CD40-CD40
ligand (CD40L) interaction using human Fc immunoglobulin G1 (IgG1)-mouse **CD40 fusion** protein (CD40-Ig); however, germinal centre (GC) formation is not. We show here that the block in B-cell differentiation in these mice is at the stage of rescue from apoptosis and exit from the GC. Thus, GC from CD40-Ig-treated mice contain a three- to fourfold higher level of apoptotic cells than found in control mice injected with human IgG1 alone. This increase in apoptosis is not caused

by a blockade of the CD40L-mediated rescue signal but is the result of an intrinsic defect of GC B cells in CD40-Ig-treated mice to receive rescue signals via CD40. While anti-CD40 stimulation maintained the viability in culture of GC B cells from control mice, it did not rescue GC B cells from CD40-Ig-treated mice. This data is consistent with the notion that a 'rewiring' of the CD40 molecule is induced by CD40 ligation early in the response and is necessary to allow B-cell rescue from apoptosis when they subsequently enter the GC.

=> d l1 1- ibib abs
YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L1 ANSWER 1 OF 3 MEDLINE
ACCESSION NUMBER: 2001209684 MEDLINE
DOCUMENT NUMBER: 21195371 PubMed ID: 11298824
TITLE: Rewiring of CD40 is necessary for delivery of rescue signals to B cells in germinal centres and subsequent entry into the memory pool.
AUTHOR: Siepmann K; Skok J; van Essen D; Harnett M; Gray D
CORPORATE SOURCE: Department of Immunology, Imperial College School of Medicine, Hammersmith Hospital, London, UK.
SOURCE: IMMUNOLOGY, (2001 Mar) 102 (3) 263-72.
Journal code: GH7; 0374672. ISSN: 0019-2805.
PUB. COUNTRY: England: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20010517
Entered Medline: 20010510
AB Memory B-cell development is impaired by in vivo blockade of the CD40-CD40 ligand (CD40L) interaction using human Fc immunoglobulin G1 (IgG1)-mouse **CD40 fusion** protein (CD40-Ig); however, germinal centre (GC) formation is not. We show here that the block in B-cell differentiation in these mice is at the stage of rescue from apoptosis and exit from the GC. Thus, GC from CD40-Ig-treated mice contain a three- to fourfold higher level of apoptotic cells than found in control mice injected with human IgG1 alone. This increase in apoptosis is not caused by a blockade of the CD40L-mediated rescue signal but is the result of an intrinsic defect of GC B cells in CD40-Ig-treated mice to receive rescue signals via CD40. While anti-CD40 stimulation maintained the viability in culture of GC B cells from control mice, it did not rescue GC B cells from CD40-Ig-treated mice. This data is consistent with the notion that a 'rewiring' of the CD40 molecule is induced by CD40 ligation early in the response and is necessary to allow B-cell rescue from apoptosis when they subsequently enter the GC.

L1 ANSWER 2 OF 3 MEDLINE
ACCESSION NUMBER: 94275366 MEDLINE
DOCUMENT NUMBER: 94275366 PubMed ID: 7516404
TITLE: Memory B cell development but not germinal center formation

is impaired by in vivo blockade of CD40-CD40 ligand interaction.

AUTHOR: Gray D; Dullforce P; Jainandunsing S

CORPORATE SOURCE: Department of Immunology, Royal Postgraduate Medical School, Hammersmith Hospital, London, UK.

SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1994 Jul 1) 180 (1) 141-55.
Journal code: I2V; 2985109R. ISSN: 0022-1007.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199407

ENTRY DATE: Entered STN: 19940729
Last Updated on STN: 19960129
Entered Medline: 19940721

AB To study the role of the CD40-CD40 ligand interaction in the development of memory B cells and its level of action during primary antibody responses in vivo, mice were injected with a soluble **CD40 fusion** protein (sCD40-gamma 1), so as to block the interaction. The effects of the treatment on the primary antibody response were reminiscent of hyper-immunoglobulin M (IgM) syndrome (HIMG1): antigen-specific IgG responses were grossly inhibited whereas the IgM response was augmented severalfold. The latter observation suggests that there is a T-dependent, CD40 ligand-independent pathway of B cell activation that leads to IgM responses and that a significant component of the IgM in HIMG1 patients is derived from T-dependent responses. The secondary response was not readily blocked by sCD40-gamma 1 treatment, indicating a relative independence of CD40 ligation of antigen-experienced B cells. The most striking finding from these studies is that the development of memory B cell populations (measured by adoptive transfer) is grossly impaired by administration of sCD40-gamma 1 during the early induction phase of the response. It is surprising that although the generation memory is diminished, there is no quantitative difference in the development of germinal centers. Whereas entry of B cells into the memory cell pathway is dependent on CD40 ligation, the clonal expansion of the potential memory precursors in germinal centers seems not to require a CD40 signal.

L1 ANSWER 3 OF 3 MEDLINE

ACCESSION NUMBER: 94267169 MEDLINE

DOCUMENT NUMBER: 94267169 PubMed ID: 7515910

TITLE: Costimulation through CD28 enhances T cell-dependent B cell activation via CD40-CD40L interaction.

AUTHOR: Klaus S J; Pinchuk L M; Ochs H D; Law C L; Fanslow W C; Armitage R J; Clark E A

CORPORATE SOURCE: Department of Microbiology, University of Washington, Seattle 98195.

CONTRACT NUMBER: DE 08229 (NIDCR)
GM 38905 (NIGMS)
RR 00166 (NCRR)
+

SOURCE: JOURNAL OF IMMUNOLOGY, (1994 Jun 15) 152 (12) 5643-52.

Journal code: IFB; 2985117R. ISSN: 0022-1767.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199407
ENTRY DATE: Entered STN: 19940721
Last Updated on STN: 20000303
Entered Medline: 19940713

AB Changes in T cell helper function were analyzed when anti-CD3-activated T cells were costimulated with mAbs to the CD28 receptor (anti-CD28). T cell-dependent B cell growth and differentiation were consistently augmented if anti-CD3 stimulated-T cells were simultaneously activated with anti-CD28. Although anti-CD28 enhanced IL-2 and IL-4 production, it did not increase B cell responses solely by augmenting production of soluble lymphokines. Anti-CD28 costimulation induced increases on T cells of CD40 ligand (CD40L), known to promote B cell proliferation and Ig secretion. Because anti-CD28 promoted T cell helper functions and expression of CD40L, we examined the dependence for CD40L during T cell-dependent B cell responses. Although soluble **CD40 fusion** proteins only partially inhibited T cell-dependent B cell activation, we found a strict requirement for CD40L expression at initiating B cell responses. Both CD40L expression and T cell help were blocked by cyclosporin A after TCR cross-linking, and, unlike T cell proliferation, both remained cyclosporin A sensitive during CD28 costimulation. In addition, anti-CD28 could not compensate for the T cell helper deficiency of hyper IgM syndrome patients who lack functional CD40L. Thus, anti-CD28-induced T cell help is delivered via a CD40L-dependent process. The fact that cross-linking CD40 on B cells promotes expression of the B7/BB-1 ligand for CD28 suggest T and B interactions may have a reciprocal amplification mechanism.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.00	2.15

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NEWS	5	Apr 23	Search Derwent WPINDEX by chemical structure
NEWS	6	Apr 23	PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
NEWS	7	May 07	DGENE Reload
NEWS	8	Jun 20	Published patent applications (A1) are now in USPATFULL
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NEWS EXPRESS	July 11 CURRENT WINDOWS VERSION IS V6.0b, CURRENT MACINTOSH VERSION IS V5.0C (ENG) AND V5.0JB (JP), AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2001
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FILE 'HOME' ENTERED AT 16:58:40 ON 06 AUG 2001

=> file medline caplus

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.15	0.15

FILE 'MEDLINE' ENTERED AT 16:58:57 ON 06 AUG 2001

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=> s tumor necrosis factor superfamily or TNFSF
L1 60 TUMOR NECROSIS FACTOR SUPERFAMILY OR TNFSF

=> s CD40L or CD154
L2 2368 CD40L OR CD154

=> s l1 and l2
L3 8 L1 AND L2

=> duplicate remove
ENTER L# LIST OR (END):l3
DUPLICATE PREFERENCE IS 'MEDLINE, CAPLUS'
KEEP DUPLICATES FROM MORE THAN ONE FILE? Y/(N):n
PROCESSING COMPLETED FOR L3
L4 6 DUPLICATE REMOVE L3 (2 DUPLICATES REMOVED)

=> d l4 1- ibib, abs
YOU HAVE REQUESTED DATA FROM 6 ANSWERS - CONTINUE? Y/(N):y

L4 ANSWER 1 OF 6 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2001:435124 CAPLUS
DOCUMENT NUMBER: 135:45182
TITLE: Multimeric forms of TNF superfamily ligands
INVENTOR(S): Kornbluth, Richard S.
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 73 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2001042298	A1	20010614	WO 2000-US7380	20000320

W: AU, CA, JP
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
PRIORITY APPLN. INFO.: US 1999-454223 A 19991209
AB A method for constructing stable bioactive fusion proteins of the difficult to express **tumor necrosis factor superfamily** (**TNFSF**), and particularly members **CD40L** (**CD154**) and RANKL/TRANCE, with collectins, particularly pulmonary surfactant protein D (SPD) is described. Single trimers of these proteins lack the full stimulatory efficacy of the natural membrane forms of these proteins in many cases. The multimeric nature of these sol. fusion proteins enables them to engage multiple receptors on the responding cells, thereby, mimicking the effects of the membrane forms of these ligands. For **CD40L**-SPD, the resulting protein stimulates B cells, macrophages, and dendritic cells, indicating its potential usefulness as a vaccine adjuvant. The large size of these fusion proteins makes them less likely to diffuse into the circulation, thereby limiting their potential systemic toxicity. This property may be esp. useful when these proteins are injected locally as a vaccine adjuvant

or tumor immunotherapy agent to prevent them from diffusing away. In addn., these and other **TNFSF**-collecting fusion proteins present new possibilities for the expression of highly active, multimeric, sol. **TNFSF** members.

REFERENCE COUNT: 2

REFERENCE(S): (1) Gires, O; EMBO J 1999, V16(20), P6131
(2) Pison, U; Eur J Clin Inv 1994, V24(9), P586

CAPLUS

L4 ANSWER 2 OF 6 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER: 2000:505293 CAPLUS

DOCUMENT NUMBER: 133:191263

TITLE: Analysis of TNF-receptor and ligand superfamily molecules in patients with lymphoproliferative disease of granular lymphocytes

AUTHOR(S): Zambello, Renato; Trentin, Livio; Facco, Monica; Siviero, Marta; Galvan, Silvia; Piazza, Francesco; Perin, Alessandra; Agostini, Carlo; Semenzato, Gianpietro

CORPORATE SOURCE: Division of Hematology, Vicenza Hospital, Vicenza, Italy

SOURCE: Blood (2000), 96(2), 647-654
CODEN: BLOOAW; ISSN: 0006-4971

PUBLISHER: American Society of Hematology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB In 21 patients with lymphoproliferative disease of granular lymphocytes (LDGL), the authors investigated the expression and the function of mols. belonging to TNF-receptor and TNF-ligand superfamilies (CD30/CD30L; CD40/**CD40L**; CD27/CD70; Fas [CD95]/FasL [CD95L]). Fourteen patients were characterized by a proliferation of granular lymphocytes (GLs) expressing the CD3+CD16+ phenotype, whereas 7 cases showed the CD3-CD16+ CD56.+-. phenotype. The data show that both CD3+ and CD3-GLs are preferentially equipped with CD30, CD40, **CD40L**, CD70, and CD95 antigens; this pattern is usually assocd. with the lack of CD27 and CD30L antigens expression. CD95L was demonstrated in the cytoplasm in 14 of 21 cases by flow cytometry, but a definite signal was demonstrated in all cases studied using polymerase chain reaction anal. On functional grounds, a stimulatory activity on rIL-2 mediated redirected-cytotoxicity against Fc.gamma.+ P815 targets was demonstrated with anti-CD30, CD40, **CD40L**, CD70, CD95, and CD95L mAbs, although resting cells were unable to exhibit significant redirected-cell lysis. The addn. of anti-CD30, CD30L, CD40, **CD40L**, CD95, and CD95L mAbs did not show any significant effect on cell proliferation at resting conditions or after rIL-2 stimulation, whereas anti-CD70 mAb mediated cell proliferation in 6 of 10 cases tested. This figure was not related to an increase in apoptotic cells, as investigated by annexin-V expression. The data indicate that both CD3+ and CD3- GLs are equipped with different costimulatory antigens, supporting the concept that these cells are in vivo activated and suggesting that these mols. might play a role in the cytotoxic mechanisms of GLs.

REFERENCE COUNT: 42

REFERENCE(S): (1) Agrawal, B; J Immunol 1996, V157, P3229 CAPLUS
(2) Alderson, M; J Exp Med 1993, V178, P2231 CAPLUS
(3) Arase, H; J Exp Med 1995, V181, P1235 CAPLUS
(4) Armant, M; Eur J Immunol 1996, V26, P1430 CAPLUS

(5) Armitage, R; Curr Opin Immunol 1994, V6, P407
CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L4 ANSWER 3 OF 6 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1998367575 MEDLINE
DOCUMENT NUMBER: 98367575 PubMed ID: 9682002
TITLE: Glucocorticoids inhibit CD40 ligand expression of peripheral CD4+ lymphocytes.
AUTHOR: Bischof F; Melms A
CORPORATE SOURCE: Department of Neurology, Eberhardt Karls University, Tubingen, Germany.. Felix.Bischof@uni-tuebingen.de
SOURCE: CELLULAR IMMUNOLOGY, (1998 Jul 10) 187 (1) 38-44.
Journal code: CQ9; 1246405. ISSN: 0008-8749.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199808
ENTRY DATE: Entered STN: 19980828
Last Updated on STN: 19980828
Entered Medline: 19980820

AB The ligand for CD40 (**CD40L**) is a type II transmembrane glycoprotein that belongs to the **tumor necrosis factor superfamily**. **CD40L** expression on peripheral CD4+ cells is increased upon activation and delivers signals to B lymphocytes which constitutively express CD40. We show that dexamethasone in vitro inhibits **CD40L** expression in a dose-dependent manner in concentrations ranging from 0.1 to 1 mg/mL. Semiquantitative analysis of **CD40L** mRNA by RT-PCR revealed that this effect was due to inhibition of **CD40L** transcription. The inhibitory effect of dexamethasone on **CD40L** expression was reversible and not due to affection of cell viability. Lymphocytes which have been exposed to dexamethasone in vitro retained the ability to express **CD40L** after incubation in medium alone for 48 h. Dexamethasone also inhibited PMA/ionomycin induced IL-2 and IFN-gamma production but not CD25 and CD69 expression. Glucocorticoids may exert their immunosuppressive effect in part by suppression of **CD40L**. Regulation of **CD40L** expression is steroid sensitive and may be similar or in part identical with IL-2 and IFN-gamma regulation.

L4 ANSWER 4 OF 6 MEDLINE
ACCESSION NUMBER: 97061417 MEDLINE
DOCUMENT NUMBER: 97061417 PubMed ID: 8905447
TITLE: Molecular, structural, and biological characteristics of the tumor necrosis factor ligand superfamily.
AUTHOR: Gruss H J
CORPORATE SOURCE: Department of Internal Medicine III, University of Ulm Medical Center, Germany.
SOURCE: INTERNATIONAL JOURNAL OF CLINICAL AND LABORATORY RESEARCH, (1996) 26 (3) 143-59. Ref: 238
Journal code: A81; 9206491. ISSN: 0940-5437.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970321
Last Updated on STN: 19980206
Entered Medline: 19970310

AB The tumor necrosis factor receptor superfamily at present consists of ten different transmembrane (type I) glycoproteins with characteristic limited sequence homology for the cysteine-rich repeats in the extracellular domain. In parallel the tumor necrosis factor ligand super-family has been recognized by discovery of ligands for all members of the receptor superfamily. These molecules are also transmembrane (type II) glycoproteins, with the exception of lymphotoxin-alpha which is the only entirely secreted protein of the tumor necrosis factor-like proteins. Several members of the ligand superfamily, including tumor necrosis factor and CD95L also exist in a biologically active soluble form. The tumor necrosis factor ligand superfamily contains at present ten different proteins. In addition, NGFR p75 binds to a second family of proteins (neurotrophins). These nerve growth factor-like dimeric soluble molecules are basic neurotrophic factors and the five members (NGF, BDNF, NT-3, NT-4, NT-5) are not related to the **tumor necrosis factor superfamily** ligands. The members of the tumor necrosis factor ligand superfamily (TNF, LT-alpha, LT-beta, CD27L, CD30L, **CD40L**, CD95L, 4-IBB, OX40L, TRAIL) share common biological activities, but some properties are shared by only some ligands, while others are unique. The diverse biological activities triggered through tumor necrosis factor receptors have been linked to the regulation of cellular activation, including immune responses and inflammatory reactions, but also with the pathology of a series of human diseases.

L4 ANSWER 5 OF 6 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 96062032 MEDLINE
DOCUMENT NUMBER: 96062032 PubMed ID: 7589079
TITLE: Altered CD40 ligand induction in tolerant T lymphocytes.
AUTHOR: Bowen F; Haluskey J; Quill H
CORPORATE SOURCE: Department of Pathology and Laboratory Medicine, University of Pennsylvania School of Medicine, Philadelphia, USA.
CONTRACT NUMBER: AI31569 (NIAID)
SOURCE: EUROPEAN JOURNAL OF IMMUNOLOGY, (1995 Oct) 25 (10) 2830-4. Journal code: EN5; 1273201. ISSN: 0014-2980.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199512
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19960124
Entered Medline: 19951212

AB CD40 ligand (**CD40L**) is a member of the **tumor necrosis factor superfamily** and is expressed on the surface of activated T lymphocytes. The interaction of **CD40L** with CD40 on B cells results in B cell activation, immunoglobulin (Ig) secretion and Ig class switching. To study anergy as a mechanism of murine CD4 T cell tolerance, we determined both in vivo and

in vitro that CD3-activated anergic cells are deficient in the ability to stimulate B cell proliferation, and that anergic cells are defective for the T cell receptor/CD3-mediated induction of **CD40L** expression. These results have implications for the recruitment of B cell responses by anergic T cells in vivo.

L4 ANSWER 6 OF 6 CAPLUS COPYRIGHT 2001 ACS

ACCESSION NUMBER:	1993:623852 CAPLUS
DOCUMENT NUMBER:	119:223852
TITLE:	CD30 antigen, a marker for Hodgkin's lymphoma, is a receptor whose ligand defines an emerging family of cytokines with homology to TNF
AUTHOR(S):	Smith, Craig A.; Gruss, Hans Juergen; Davis, Terri; Anderson, Dirk; Farrah, Terry; Baker, Elizabeth; Sutherland, Grant R.; Brannan, Camilynn I.; Copeland, Neal G.; et al.
CORPORATE SOURCE:	Immunex Res. and Dev. Corp., Seattle, WA, 98101, USA
SOURCE:	Cell (Cambridge, Mass.) (1993), 73(7), 1349-60 CODEN: CELLB5; ISSN: 0092-8674
DOCUMENT TYPE:	Journal
LANGUAGE:	English

AB CD30 is a surface marker for neoplastic cells of Hodgkin's lymphoma and shows sequence homol. to members of the tumor necrosis factor (TNF) receptor superfamily. Using a chimeric probe consisting of the extracellular domain of CD30 fused to truncated Ig heavy chains, the cDNA cognate from the murine T cell clone 7B9 was expression cloned. The encoded protein is a 239 amino acid type II membrane protein whose C-terminal domain shows significant homol. to TNF.alpha., TNF.beta., and **CD40L**. Cross-hybridization to an induced peripheral blood T cell cDNA library yielded the human homolog, which is 72% identical at the amino acid level. The recombinant human ligand enhances the proliferation of CD3-activated T cells yet induces differential responses, including cell death, in several CD30+ lymphoma-derived clones. The human and murine genes map to 9q33 and the proximal region of chromosome 4, resp.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	19.88	20.03
DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-1.76	-1.76

STN INTERNATIONAL LOGOFF AT 17:02:21 ON 06 AUG 2001